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Brief report

A locus in *Drosophila melanogaster* affecting heat resistance

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Temperature is an important environmental factor for *Drosophila melanogaster* and polymorphism for temperature resistance is very likely to occur (PARSONS 1973). Many studies are published about heat resistance in *Drosophila melanogaster,* but most of them concern the heat shock response (e.g., LINDQUIST 1986). Little attention is paid to the variation in heat resistance itself, though such studies can reveal additional information about the mechanisms of heat resistance and climatic adaptations. Above this, conditional lethal mutations can play a role in agricultural pest control (e.g., HEDRICK 1984).

The first report of variability for heat resistance in *Drosophila melanogaster* is from HOSGOOD and PARSONS (1968), who found differences between iso-female lines and ascribed these to (unknown) polymorphic genes. MORRISON and MILKMAN (1978) succeeded in selection for decreased resistance, and showed that the gene or genes responsible for this decrease could be localized for the greater part at the second chromosome. The present article describes the genetical localization of a natural mutant for decreased heat resistance in *Drosophila melanogaster*: a recessive heat sensitive lethal on the second chromosome, *l(2)hs*.

Experiments

For all experiments the flies were reared at 25°C and about 50 % R.H. on standard medium (OUDMAN et al. 1991) and tests were performed at 35°C and about 90 % R.H. to prevent desiccation.

The mutant was discovered in the wild type laboratory strain Groningen 83, which was founded in 1983 with 403 females from a fruit market in Groningen, the Netherlands. Fig. 1 shows survival at 35°C of males of a substrain that was extracted from Groningen 83 for other purposes (OUDMAN et al., in preparation). The figure clearly shows two phases of mortality, one during the first 18 hours and one starting at 48 hours. Other strains tested at

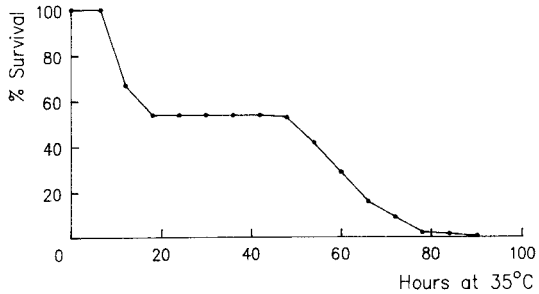

Fig. 1. Survival at 35°C of males from a polymorphic strain of *Drosophila melanogaster*.

35°C (not shown) did not have two phases of mortality, but had normal curves with mortality starting at about 48 hours. When survival at 25°C was followed from a strain that had two mortality phases at 35°C, a normal survival curve was observed (Fig. 2), as was the fact at 29°C (not shown). Thus, the two phases of mortality at 35°C seemed to be caused by a polymorphism for survival at high temperature that did not influence longevity at lower temperatures.

For further analysis eight inbred strains were derived from the substrain mentioned above by at least four subsequent sister–brother crosses, and tested for survival at 35°C. Table 1 shows that two strains (1 and 7) had strongly decreased heat resistance compared to the other strains. This effect was similar for males and females. In a resistant strain some flies might die during the first day, due to 'normal' mortality, as would be the fact at any other temperature. Because comparison of these strains in later generations gave similar results (not shown), it was concluded that the inbred strains were true-breeding for a genetic factor determining heat resistance.

A cross between a sensitive and a resistant strain (not shown) yielded a resistant strain; thus sensitivity was a recessive trait. Chromosome analysis was performed with the aid of the balanced marker stock

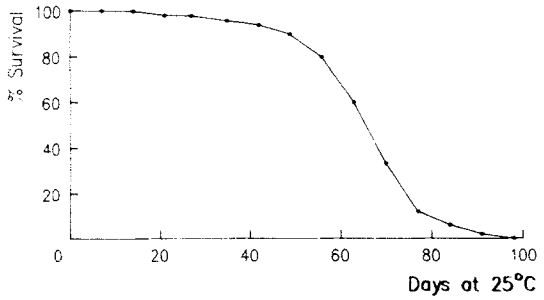

Fig. 2. Survival at 25°C of males from a polymorphic strain of *Drosophila melanogaster*.

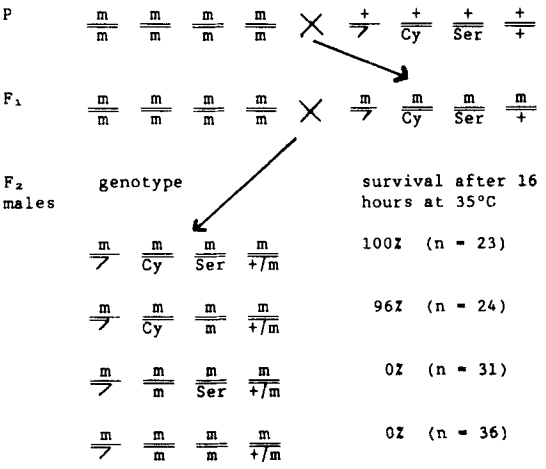

Fig. 3. Crossing scheme and survival percentages of the adult males in the F_2 of the chromosome localization experiment. (m = chromosome of heat sensitive strain).

for the second and third chromosome SM5 *Cy*: TM3 *Ser* (LINDSLEY and GRELL 1968). The observation that homozygous sensitive adults never survived for longer than 16 hours at 35°C, while resistant flies, if in good condition, normally survived much longer, was used in the chromosome localization to infer the resistance of flies. The heat sensitive strain 7 was crossed with the marker strain according to the scheme in Fig. 3. F_2 males were tested for survival during 16 hours at 35°C. From the survival percentages in Fig. 3 it can be deduced that heat sensitivity is a recessive character located on the second chromosome.

Localization experiments with the recessive mutants *cinnabar* (*cn*, II 57.5) (all standard locations according to LINDSLEY and GRELL 1968) and *brown* (*bw*, II 104.5) (not shown) induced that the character was located just left of *cn*. Final localization on the second chromosome was performed with the recessive mutants *purple* (*pr*, II 54.5) and *cn*. Females of the heat sensitive strain 7 were crossed with males *pr cn*. F_2 females (heterozygotes in which recombination could occur) were backcrossed to *pr cn* males. In the F_2 four phenotypes occurred: wild type, purple, cinnabar, and purple/cinnabar (Table 2). In the F_2 a recombination percentage between *pr* and *cn* of 2.3 % was found, instead of the expected 3 %, which is significantly different ($\chi^2_1 = 3.96$, $0.04<P<0.05$). This difference might be caused by different viability of the recombinant genotypes, but there were no other indications for such differences. To determine the frequency of sensitivity in the F_2 phenotypes, a number of individual males from each of the F_2 phenotypes were crossed with sensitive females from strain 7, and their progeny were kept apart. These F_3 flies were tested for survival at 35°C. If sensitivity was present in an F_2 male in heterozygous state, mortality of F_3 progeny was expected to be 50 %. If sensitivity was not present in the F_2 male, 100 % survival of F_3 progeny was expected. At least 20 flies per cross were tested at 35°C. Table 2 shows the number of the F_2 phenotypes and the results of the F_3 tests. Because the ratio sensitive/non-sensitive was reversed between the recombinant phenotypes (*pr* and *cn*) and between the non-recombinant phenotypes (wild type and *pr cn*), ap-

Table 1. Percentage adult mortality after various periods of time at 35°C in eight inbred strains of *Drosophila melanogaster* (n individuals tested)

Strain	Females, hours at 35°C					Males, hours at 35°C				
	12	24	36	48	(n)	12	24	36	48	(n)
1	100	100	100	100	(80)	100	100	100	100	(84)
2	0	0	18	100	(80)	0	20	100	100	(80)
3	0	2	46	100	(81)	0	9	72	100	(79)
4	0	3	19	100	(80)	0	14	91	100	(79)
5	0	5	71	100	(73)	2	33	100	100	(79)
6	0	1	13	100	(79)	0	3	91	100	(80)
7	100	100	100	100	(80)	100	100	100	100	(78)
8	1	1	11	100	(80)	1	1	36	100	(80)

Table 2. Occurrence of heat sensitivity in F_3 males from crosses of F_2 males with heat sensitive females

Phenotype	Number in F_2	Number F_3 tested	Resistant	Sensitive
wild type	1233	121	0	121
cinnabar	29	27	10	17
purple	29	27	18	9
purple/cinnabar	1208	121	121	0

parently only one locus is involved. The locus is located between *pr* and *cn*, closer to *pr* than to *cn*. The location on a standard second chromosome is 54.5 + (9 + 10)/54*3 = 55.6. The locus is a conditional, heat sensitive, recessive lethal on the second chromosome: *l(2)hs*.

Conclusion and discussion

l(2)hs is a natural, heat sensitive, recessive lethal of *Drosophila melanogaster*, with map position II 55.6. Adult flies, homozygous for *l(2)hs*, never survive 16 hours at 35°C, while wild type flies, if in good condition, usually survive much longer.

No recessive heat sensitive mutants were known from the autosomes of *Drosophila melanogaster* (LINDSLEY and ZIMM 1986). SUZUKI (1970) described a number of mutations sensitive to 29°C, but these were recessive mutations on the X-chromosome and dominant lethals on the second and third chromosome. Possibly *l(2)hs* is the same locus that influenced survival in the experiments of MORRISON and MILKMAN (1978), who only did a chromosome localization.

About the mechanism we can only speculate at the present time. Enzyme inactivation or tissue- or cell damage both are possible, perhaps intermediated by the absence of heat shock protein synthesis. No heat shock protein loci are known in the region near II 55.6 (ASHBURNER and BONNER 1979). A number of mutants that influence the expression of the heat shock response are known from the second chromosome, but they are not exactly localized (PARKER-THORNBURG and BONNER 1987). Studies of the molecular and physiological mechanism of the locus and the temperature range of sensitivity will be necessary.

Up to now *l(2)hs* is only found in the Groningen 83 population. Because the mutant has not been discovered before it is probably rare in nature, but further studies on the occurrence are necessary.

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